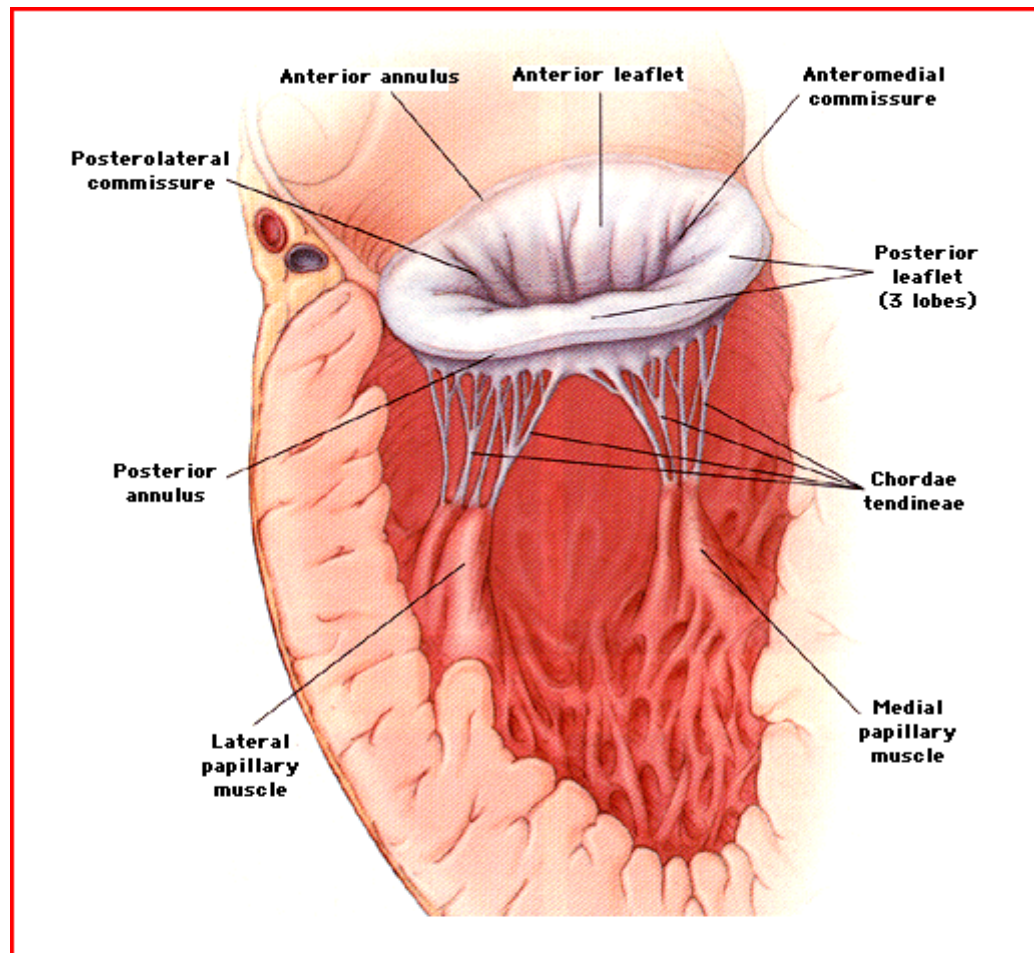
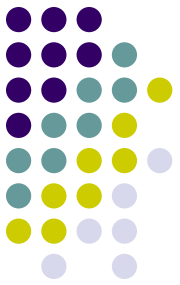


Mitral Apparatus

- Mitral Valve leaflets
- Chordae tendinae
- Papillary muscles
- MV annulus
- LV myocardium and LA wall adjacent to the Papillary muscles.





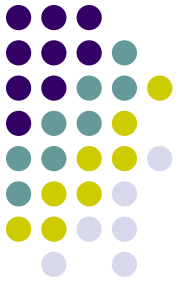
Mitral valve anatomy The mitral valve consists of the mitral annulus, anterior and posterior leaflets, chordae tendineae, and the papillary muscles. Mitral regurgitation may be due to a disease that primarily affects the valve leaflets, such as mitral-valve prolapse or rheumatic mitral-valve disease, or may result from alterations in the function or structure of the left ventricle, such as those induced by ischemic disease or dilated cardiomyopathy. (Reproduced with permission from: Otto, CM. Clinical practice. Evaluation and management of chronic mitral regurgitation. N Engl J Med 2001; 345:740. Copyright © 2001 Massachusetts Medical Society. All rights reserved.)

Incidence of Mitral Regurgitation



- Framingham study found mild MR present in 19% of men and woman (asymptomatic)
- Severe MR, dependent on the study, is present from 0.2-1.9% of the general population.

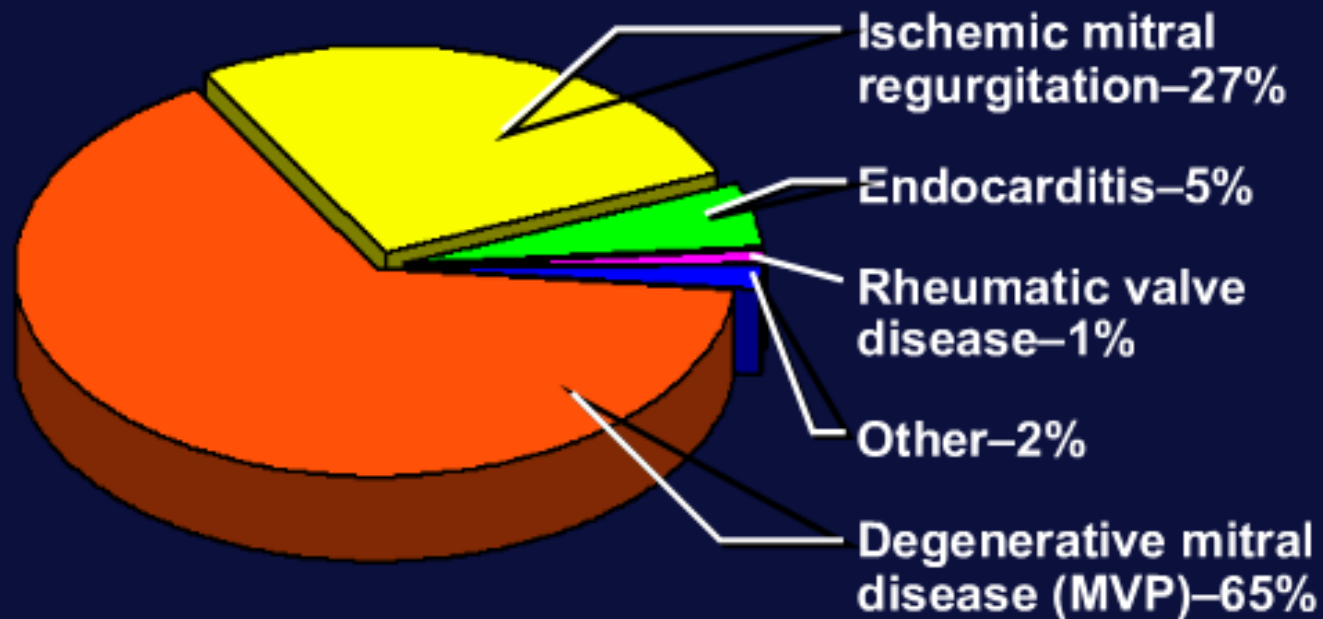
Mitral Regurgitation



1. Acute primary MR.
2. Chronic primary MR.
3. Secondary MR.



Etiology of Mitral Regurgitation



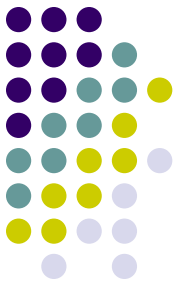
Waller BF et al: Clin Cardiol 17:395, 1994



Ischemic papillary m. dysfunction

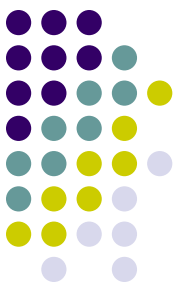


- Usually occurs with acute MI
- Can occur with anterior or inferior MI
- May occur with angina and then resolves
- Papillary m. complete rupture has a very high mortality



Ischemic MR

- About 10% of MI have MR by cath
- Moderate to severe in 3-4 %
- Some ECHO studies after MI show varying degrees of MR in +- 50 % of cases
- Old patients and woman have a higher incidence. More common with large MI
- Incidence ↓ with early reperfusion of MI
- Surgery (MVR) may be indicated and has a high morbidity and mortality.

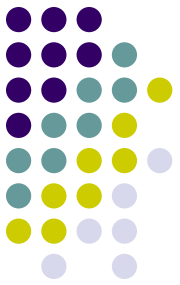


Papillary muscle rupture after MI Pathologic specimen showing complete transection of papillary muscle (arrow) after an acute myocardial infarction. The patient died with severe mitral regurgitation. (Photograph courtesy of Dr William D Edwards. From Reeder, GS, Gersh, BJ, Acute myocardial infarction. In: Internal Medicine, 4th ed, Stein, JH, Hutton, JJ, Kohler, PO, et al (Eds), Mosby-Year Book, St Louis, 1994, pp. 169-189. By permission.)



Causes of secondary MR

- CAD, old MI, ischemic cardiomyopathy
- Idiopathic dilated cardiomyopathy
- Advanced aortic valve disease
- Hypertensive heart disease
- Any cardiac disease that leads to LV dilatation



Clues that MR is secondary

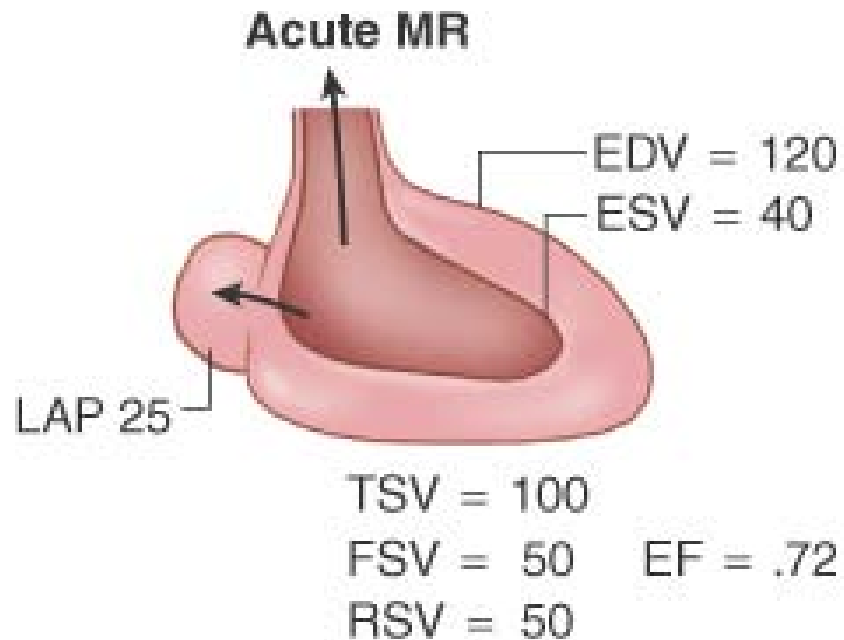
1. ECG = Severe LVH, MI.
2. Rhythm is NSR (not a fib)
3. LA is not disproportionately large
4. EF is low (uncommon in primary MR)
5. MV appears normal
6. Segmental wall motion abnormalities on Echo, MI on ECG

Common causes of acute primary MR



- Endocarditis involving the valve leaflets, cordae tendineae or annulus.
- Spontaneous (idiopathic) rupture of cordae
- Rupture of diseased cordae ie. Collagen vascular disease , Marfanoid state, RHD or MV prolapse syndrome.

Acute MR



B



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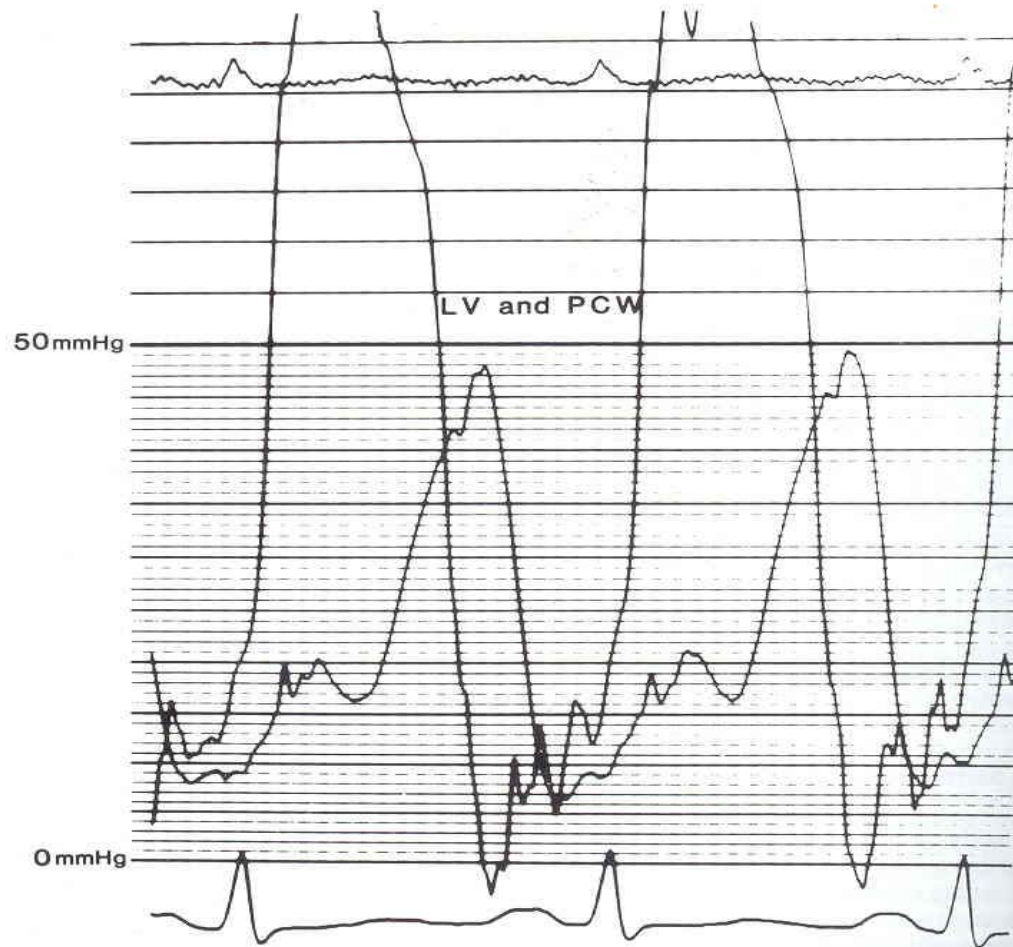
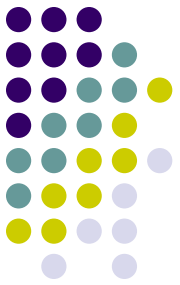
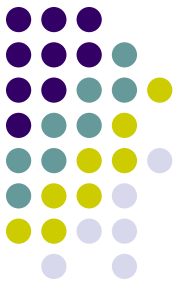
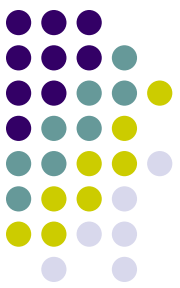


Figure 23.13. Simultaneous recording of pulmonary-capillary wedge (PCW), left ventricular (LV) pressures, and electrocardiograph in acute severe mitral regurgitation. Note the large V-wave, which frequently occurs in acute severe mitral regurgitation. The apparent early diastolic mitral valve pressure gradient is attributable to a damped pulmonary-capillary wedge tracing and rapid diastolic transmitral flow velocity, which is required by the regurgitation.

Common causes of Chronic primary MR



- Any of the causes of acute primary MR may progress to chronic primary MR
- Mitral prolapse syndrome
- Chronic RHD



Causes of Chronic Mitral Regurgitation

Leaflet

- Rheumatic fever
- Systemic lupus erythematosus
- Infective endocarditis (acute and chronic)
- Scleroderma
- Connective tissue disorders
 - Marfan's
 - Ehlers-Danlos
 - Pseudoxanthoma elasticum
- Congenital
 - Mitral valve clefts
 - Parachute mitral valve
 - Endocardial cushion defects
- Myxomatous degeneration (mitral valve prolapse)
- Left atrial myxoma
- Hypertrophic cardiomyopathy (systolic anterior movement of mitral valve)
- Fenfluramine-phentermine

Chordae tendineae

- Myxomatous degeneration (mitral valve prolapse)
- Infective endocarditis (acute and chronic)
- Trauma
- Rheumatic fever
- Rupture
 - Spontaneous
 - Myocardial infarction
 - Trauma
 - Myxomatous degeneration
 - Endocarditis

Papillary muscles

- Papillary muscle dysfunction
 - Ischemia
 - Myocardial infarction
 - Dilated cardiomyopathy
 - Left ventricular aneurysm
 - Infiltration (amyloid, granulomas)
 - Infection (endocarditis, abscess)
- Papillary muscle rupture
 - Myocardial infarction
 - Trauma

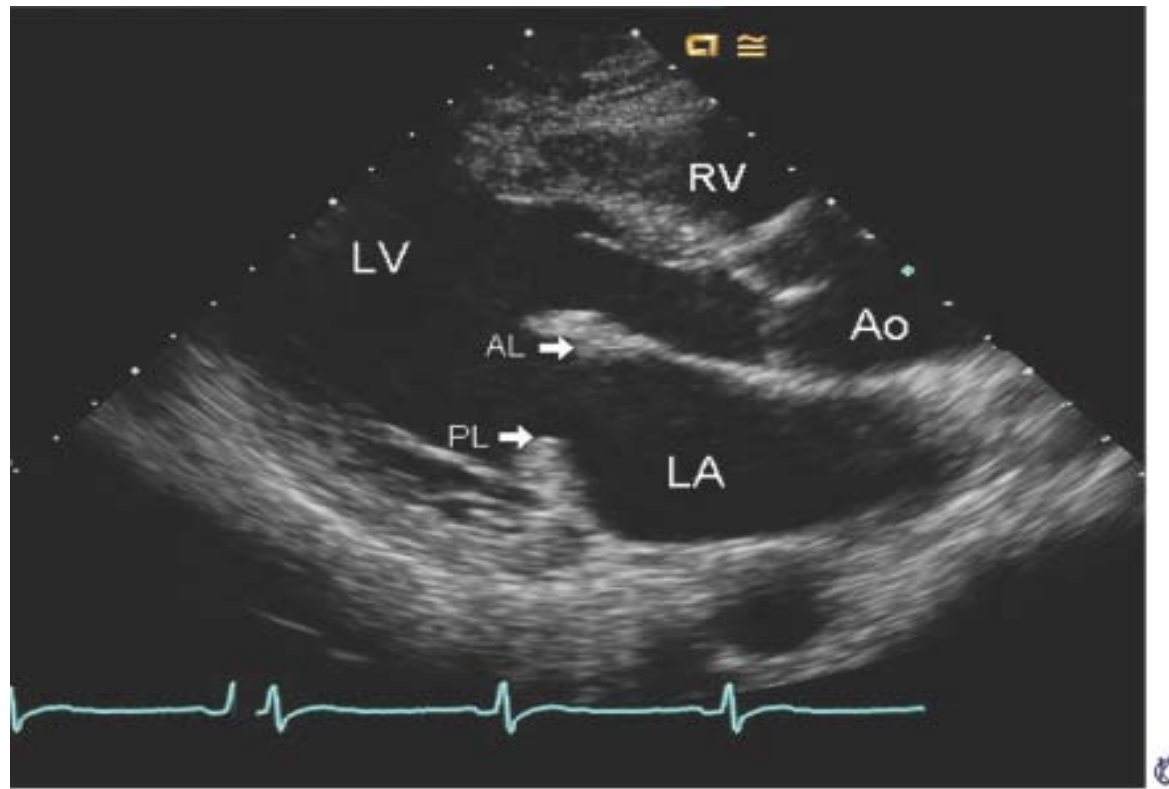
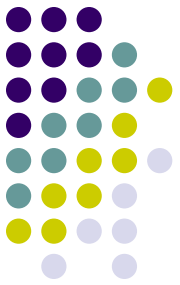
Mitral annulus

- Calcification
 - Idiopathic
 - Rheumatic fever
 - Chronic renal failure
 - Hyperparathyroidism
- Dilatation
 - Connective tissue disorder
 - Dilated cardiomyopathy

Prosthetic valve

- Paravalvular leak
- Infective endocarditis
- Ring or strut fracture
- Disc or ball dysfunction or dislodgement
- Leaflet deterioration (tissue valves)

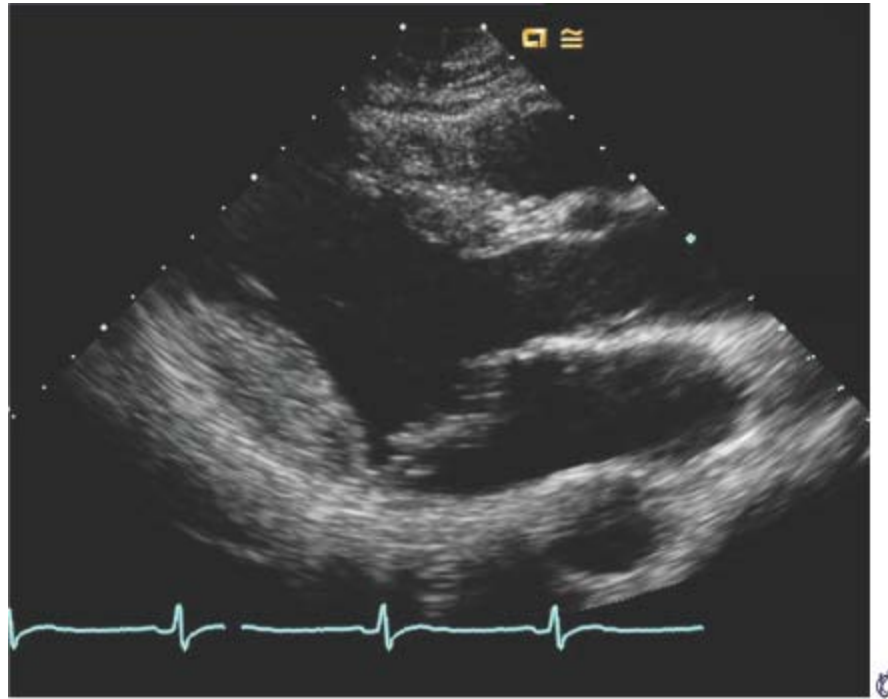
Elongated anterior MV Leaflet



A

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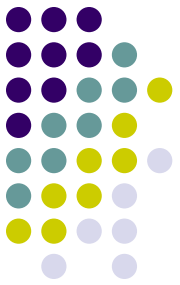
MV Prolapse



C

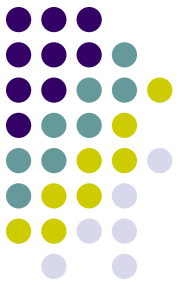
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Myxomatous MV



A

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Examples of ECHO in MR

1. RHD. MV thick and some MS
2. Endocarditis = vegetations
3. Chordal rupture = \uparrow leaflet motion
4. MVP often causes hammocking or posterior motion of leaflets.
5. Failure of leaflets to close completely suggests papillary m. dysfunction or a markedly \uparrow LV size.



TABLE 44-1 Myocardial O₂ Consumption Components*

Total

6-8 ml/min/100 gm

Distribution

Basal, 20%

Electrical, 1%

Volume work, 15%

Pressure work, 64%

Effects on MVO₂ of 50% Increase In

Wall stress, 25%

Contractility, 45%

Pressure work, 50%

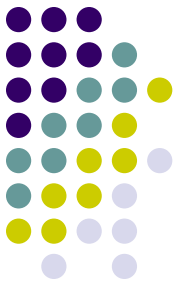
Heart rate, 50%

Volume work, 4%

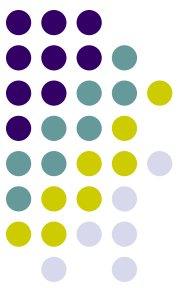
From Gould KL: Coronary Artery Stenosis. New York, Elsevier, 1991, p 8.

*The table demonstrates the dominant contribution to myocardial O₂ consumption (MVO₂) made by pressure work and prominent effects of increasing pressure work and heart rate on MVO₂.

1. Pathophysiology of Chronic MR

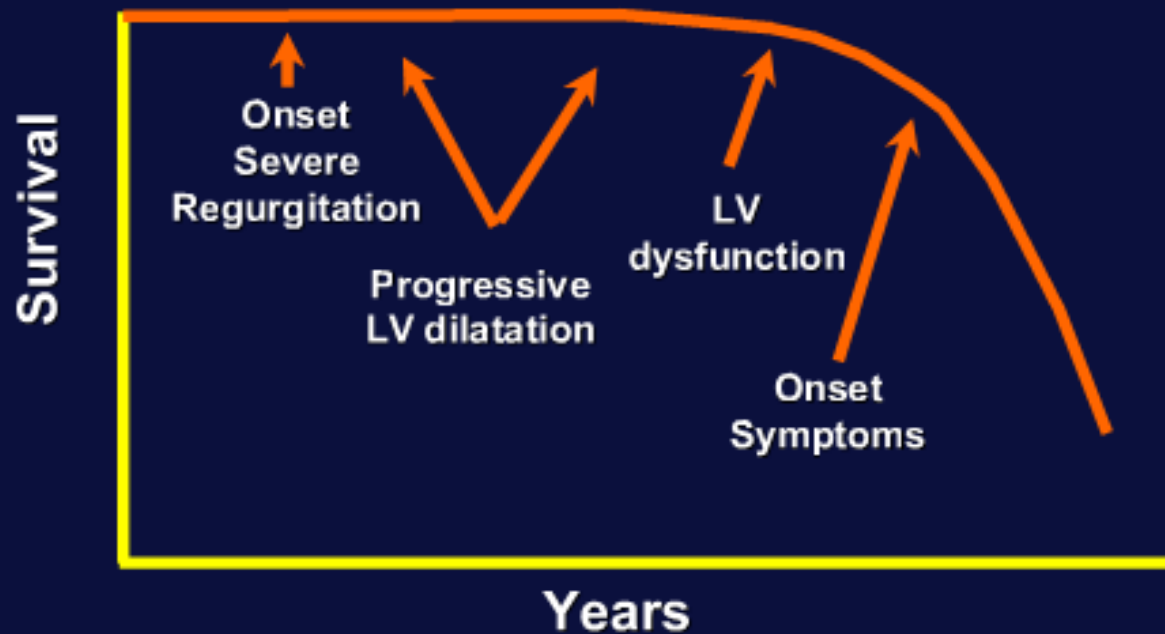


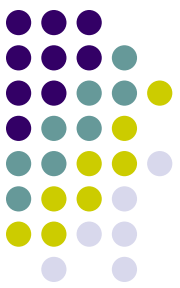
1. Isovolumetric contraction phase absent
2. Afterload greatly ↓
3. LV dilatation occurs 1st in flow loads
4. EF initially ↑ and remains high or normal
5. EF falls very late in disease
6. LA slowly dilates and may be enormous
7. CO well preserved
8. Symptoms may not occur for many years
9. Severe LVH does not occur



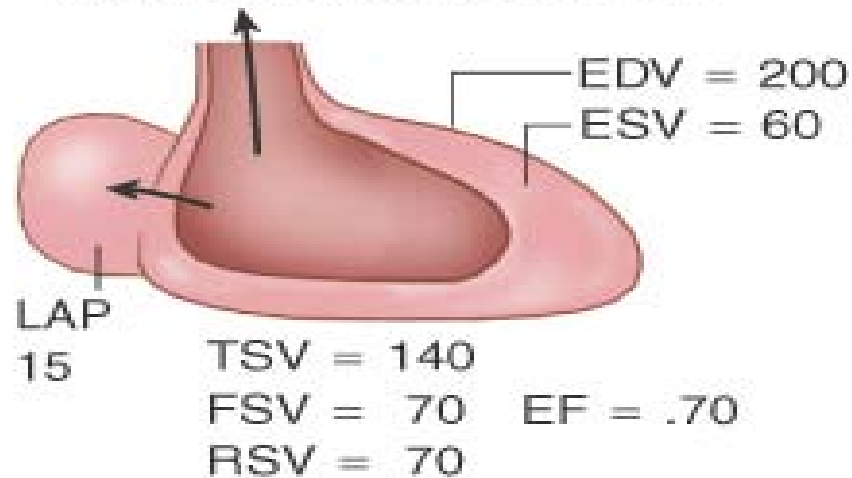
Regurgitant Lesions

Concept of Volume Overload





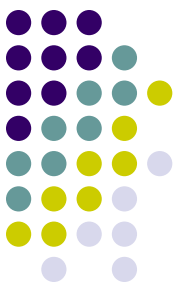
Chronic compensated MR



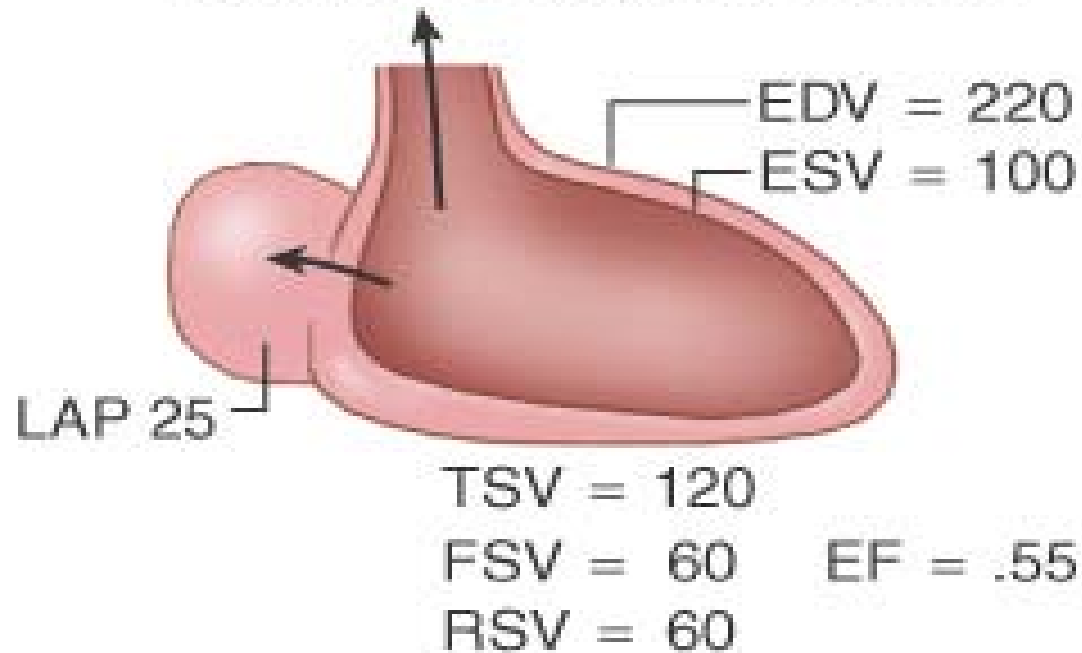
C



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Chronic decompensated MR



D

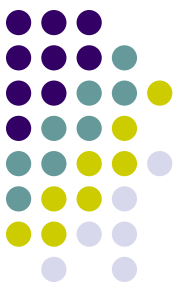


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Mitral Regurgitation Severity

- **Echo color flow alone**
Underestimate
Overestimate
- **Cath if discrepancy**



Severe Mitral Regurgitation

Echocardiographic Findings

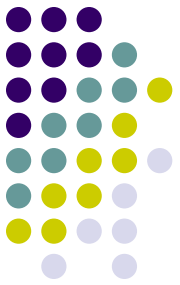
Severity

- Color jet area $>1/2$ LA area.

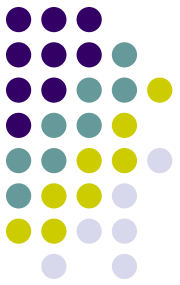
BEWARE!

- Regurgitant volume (>60 mL)
- Regurgitant fraction ($>55\%$)
- ERO (>0.4 cm²)

ECG IN CHRONIC PRIMARY MR



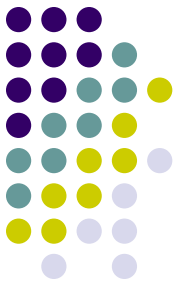
1. Atrial fibrillation is very common.
2. If in NSR P wave broadens to greater than 0.12 secs.
3. P wave be notched and have a large negative component in V1
4. If pulmonary hypertension present, RVH, tall R in V1.
5. Mild LVH (one should never see severe LVH if only Chronic MR present)..



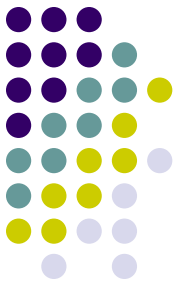
Symptoms of MR

- Symptoms are related to the decrease in forward effective cardiac output and LA pressure.
- Weakness, fatigue and poor exercise tolerance
- LV failure symptoms
- R heart failure (less common)
- Thromboembolism

Physical Exam in chronic primary MR

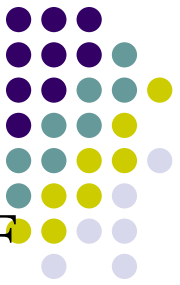


1. Arterial pulse is ↓ in volume but is brisk (reflecting high EF). Pulse pressure usually is normal-not wide as in chronic AI.
2. S1 ↓ reflecting failure of leaflets to close properly.
3. S2 may be widely split due to ↓ in LV ejection time and delay of S2P if pulmonary hypertension is present.
4. Murmur is systolic but its duration, quality, location and radiation depend on the component of the Mitral apparatus effected.
5. In most case pansystolic murmur starts with S1 and continues to S2. It is heard best at apex and radiates to axilla or back, usually blowing and high pitched in quality.



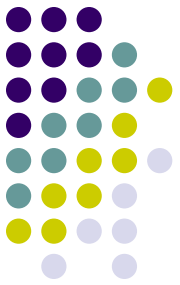
ATYPICAL MR MURMURS

1. When posterior leaflet involved due to prolapse or chordal rupture, murmur may radiate to sternum or base of heart.
2. Anterior leaflet involvement in prolapse may be very loud and radiate to neck.
3. MV prolapse M may be harsh and be mid or late systolic, preceded by a mid systolic click.
4. M of Pap. Muscle ischemia may be variable and become louder during an ischemic episode.
5. Large diastolic volumes across valve may produce a diastolic murmur.
6. Secondary MR murmur may decrease with RX of CHF.
7. No murmur, particularly if CO low



EFFECT OF DIAGNOSTIC MANEUVERS ON MR

1. MR may be louder when LV volume increases as in CHF or leg raising when supine.
2. May increase when afterload increases (squatting or isometric handgrip).
3. May be softer when venous return is decreased as in standing or Valsalva.
4. MVP murmur is longer when patient stands and click moves closer to S1.



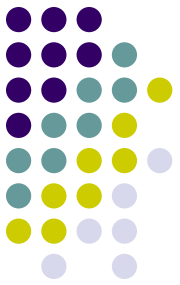
Clinical manifestations of MR.

- The nature and severity of MR symptoms depends on the:
- Amount of MR
- Rate of progression of MR
- Acute or chronic
- LA pressure(or Pa wedge pressure)
- PA pressure
- Associated cardiac disease



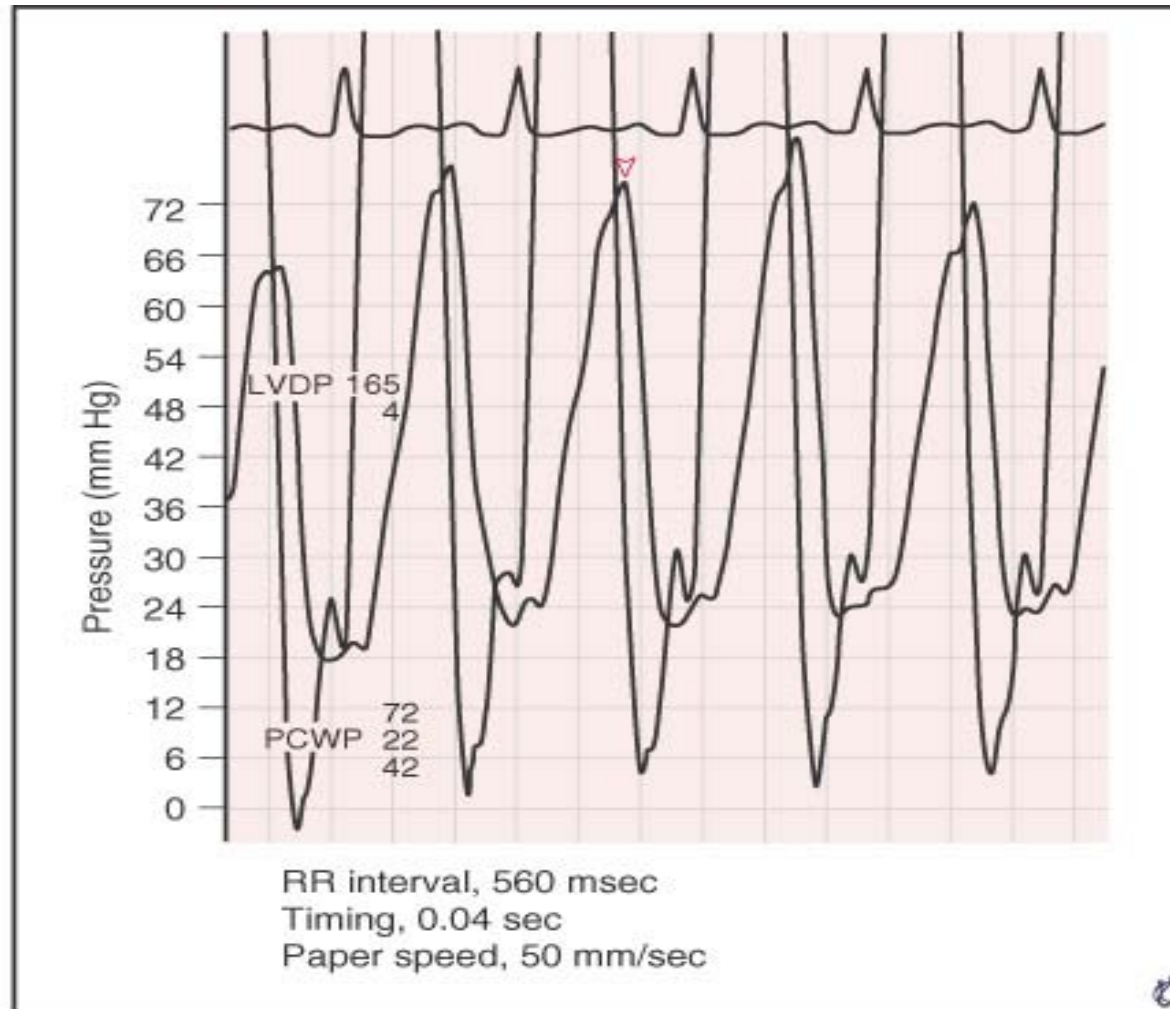
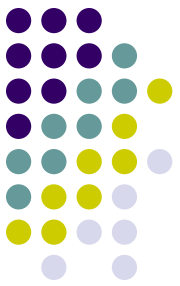
ECHOCARDIOGRAM IN MR.

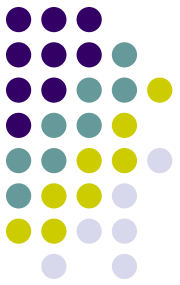
- With M mode and 2 D Echo (TTE or TEE) there is ↑ LA,
- ↑ LV and LV contraction pattern (EF ↑).
- Mechanism of MR usually identified.
- Prolapse, endocarditis, Flail leaflet, RHD etc.
- Colorflow Doppler gives degree of MR
- Degree of MR is just an estimate



Indications for TEE

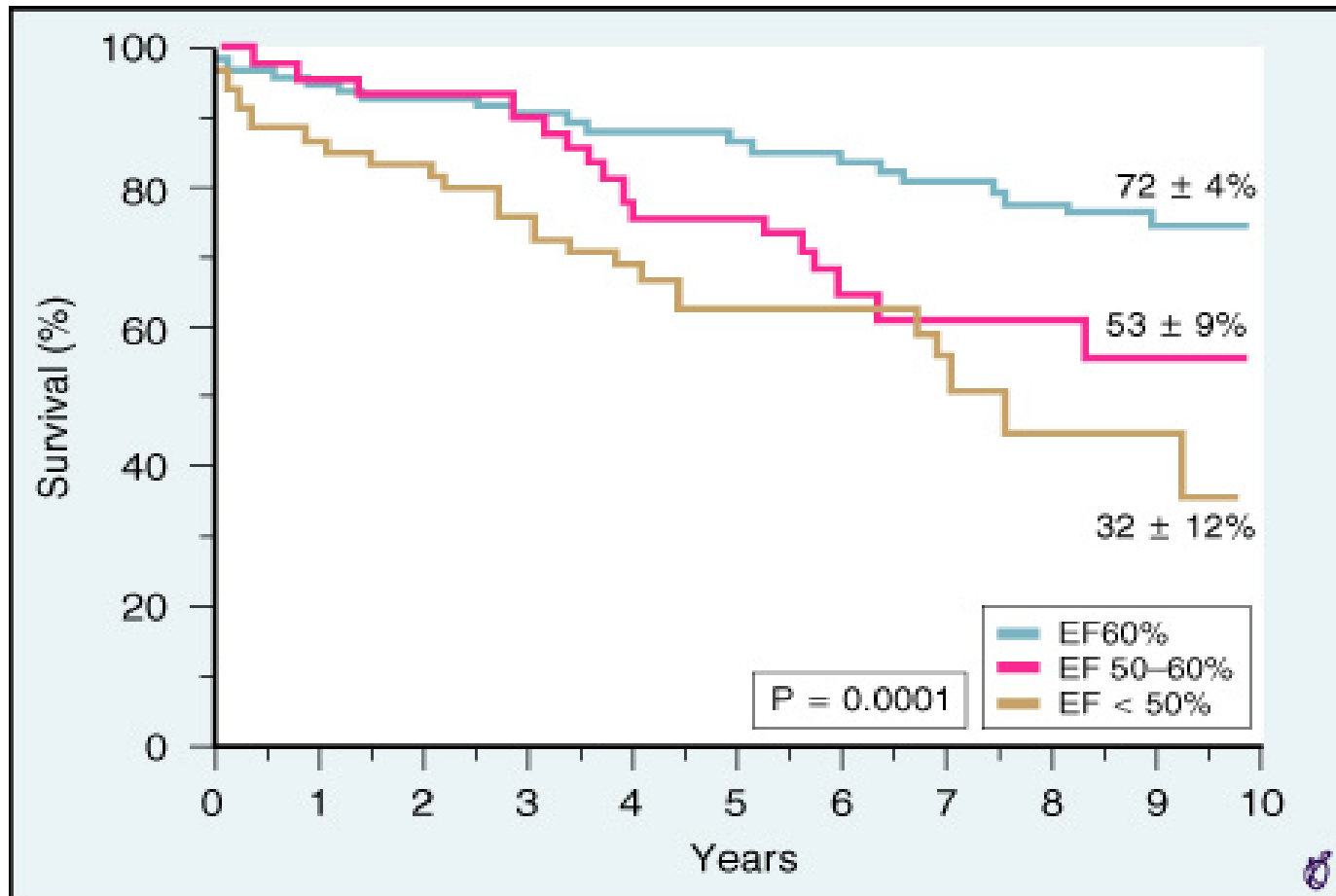
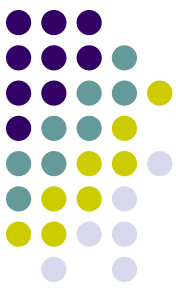
1. When TTE is of poor quality
2. When endocarditis suspected and TTE is equivocal
3. When endocarditis is suspected and TTE is negative (will sometimes see a vegetation with TEE).



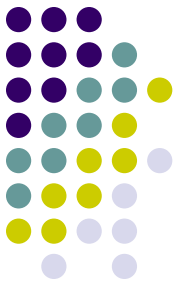


Cardiac Cath. In MR

1. Pressure recordings may show giant V waves in major acute MR.
2. Ventriculography done when Echo is poor and degree of MR uncertain
3. Coronary angiography when ischemic heart disease suspected.
4. Pressure data may be helpful to decide whether symptoms are due to MR.

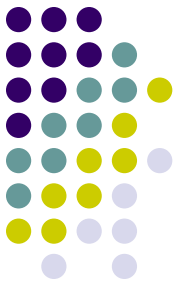


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Surgery in ACUTE MR

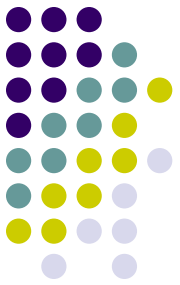
1. Torn chord and acute primary MR may cause intractable pulmonary edema requiring MVR
2. Papillary M. rupture in acute MI may require MVR if med RX fails.
3. Endocarditis with 4+ MR, surgery if patient can't be stabilized medically.



Surgery in Chronic Primary MR

1. MR may progress insidiously without symptoms.
2. If CHF occurs, surgery indicated.
3. Surgery indicated in asymptomatic MR when (55 rule applies)
4. End systolic dimension approached 55mm or EF falls below 55% (older criteria).
5. Surgery not performed in secondary MR but may be considered if MR 4+ and EF above 30%.

Indications for surgery in asymptomatic chronic MR

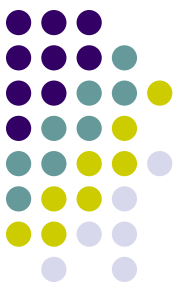


- Monitor LV size and EF
- Surgery indicated when end systolic dimension approaches 55 mm (now 40 mm)
- Surgery indicated when EF is falling and approaches 55% (now 60%)
- Some newer recommendations are based on the amount of MR. (NEJM 3/3/2005, Vol. 352:875-883) .Pts with effective regurgitant orifice $> 40\text{mm}^2$ need surgery.
- Catheter techniques for secondary MR ? Indications
- New consideration, elevated BNP levels may help in deciding whether to operate

MV SURGERY IN CHRONIC SEVERE MR (2006 guidelines) in JACC VOL.48,no.3,2006



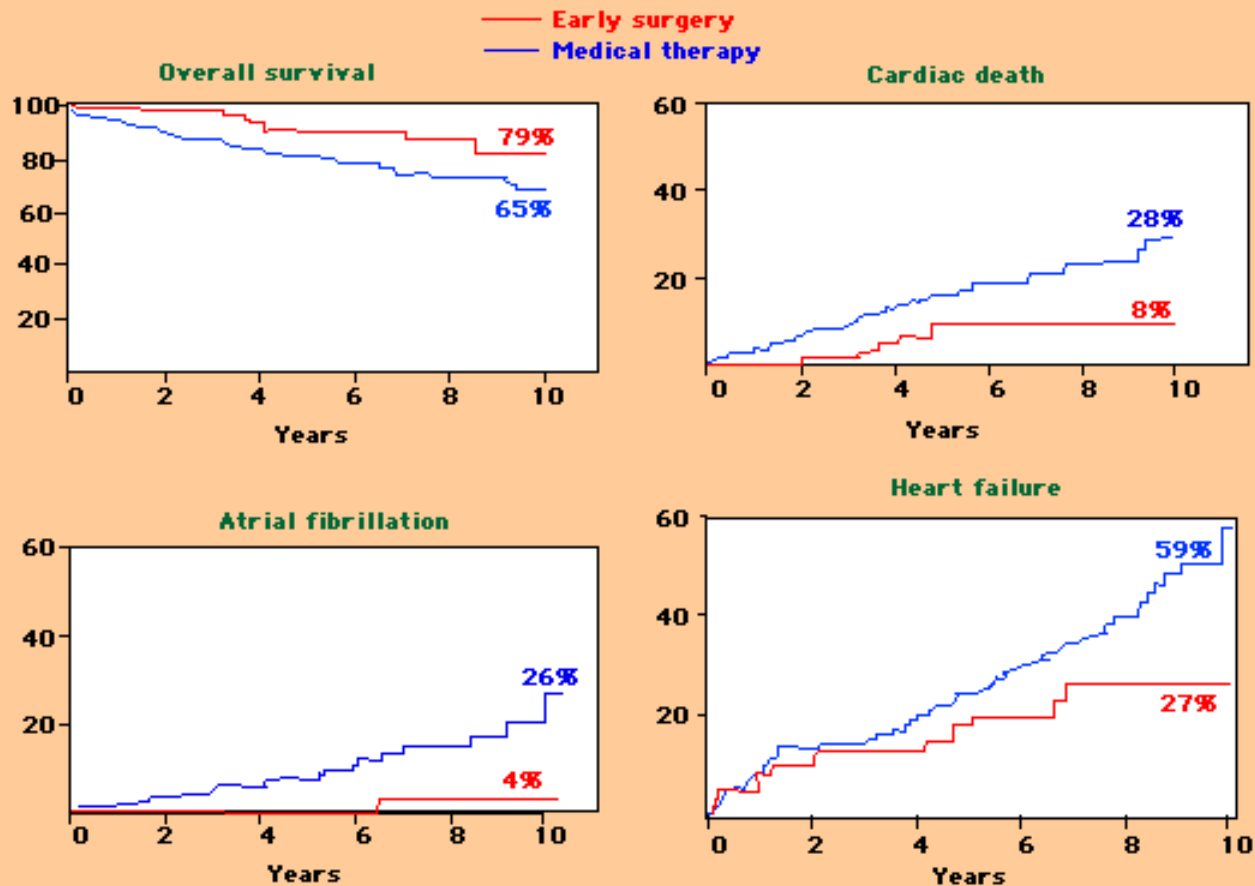
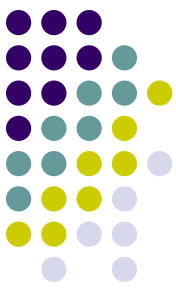
MV surgery is beneficial for asymptomatic patients with chronic severe MR and mild to moderate LV dysfunction, EF 0.30-60% and/or end-systolic dimension greater than or equal to 40mm. (level of evidence B)



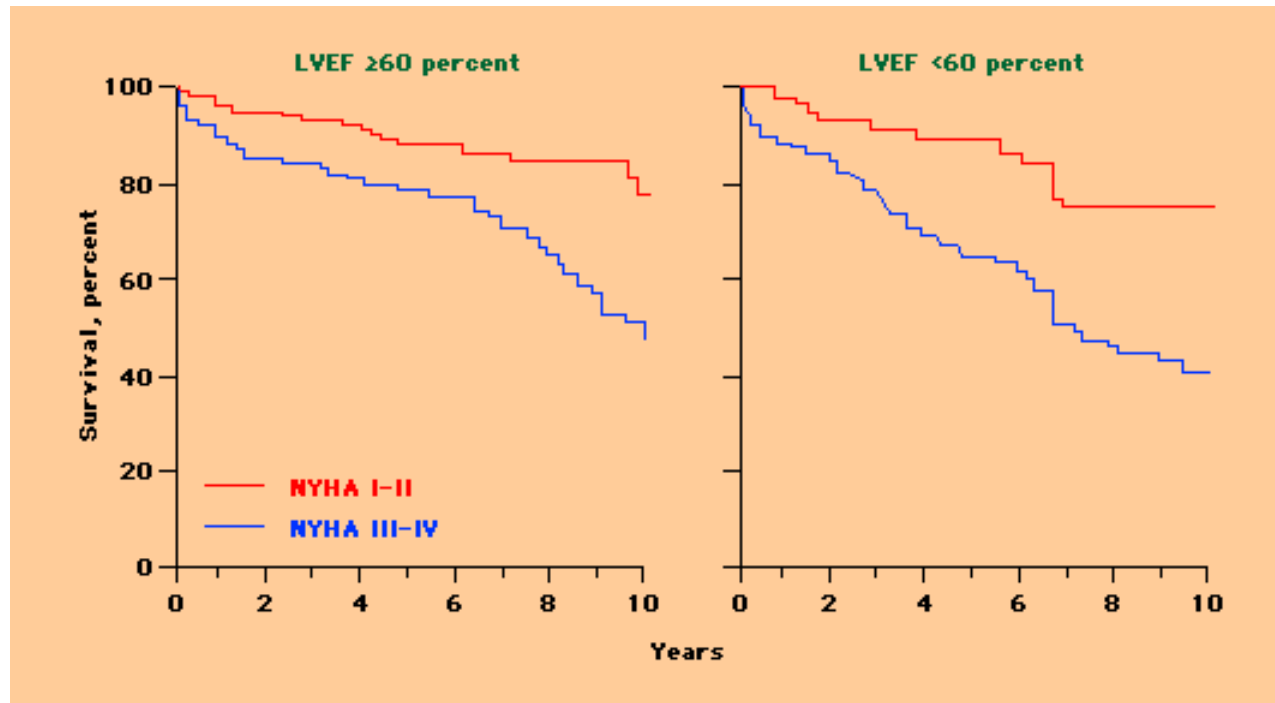
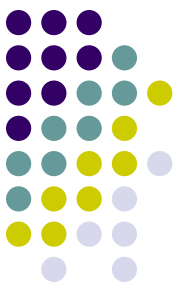
Law of Laplace

$$\text{Left ventricular wall stress} = \frac{\text{Cavity pressure} \times \text{Radius}}{\text{Wall thickness} \times 2}$$

Law of Laplace According to the law of Laplace, left ventricular (LV) wall stress is directly proportional to the product of cavity pressure and radius and inversely proportional to wall thickness. An elevation in LV cavity pressure (as with aortic stenosis or hypertension) causes an increase in wall stress. If a hypertrophic response occurs, increasing thickness can return wall stress to normal. With LV chamber enlargement (as with mitral regurgitation, dilated cardiomyopathy, or infarct expansion) wall stress increases, unless there is an appropriate hypertrophic response.

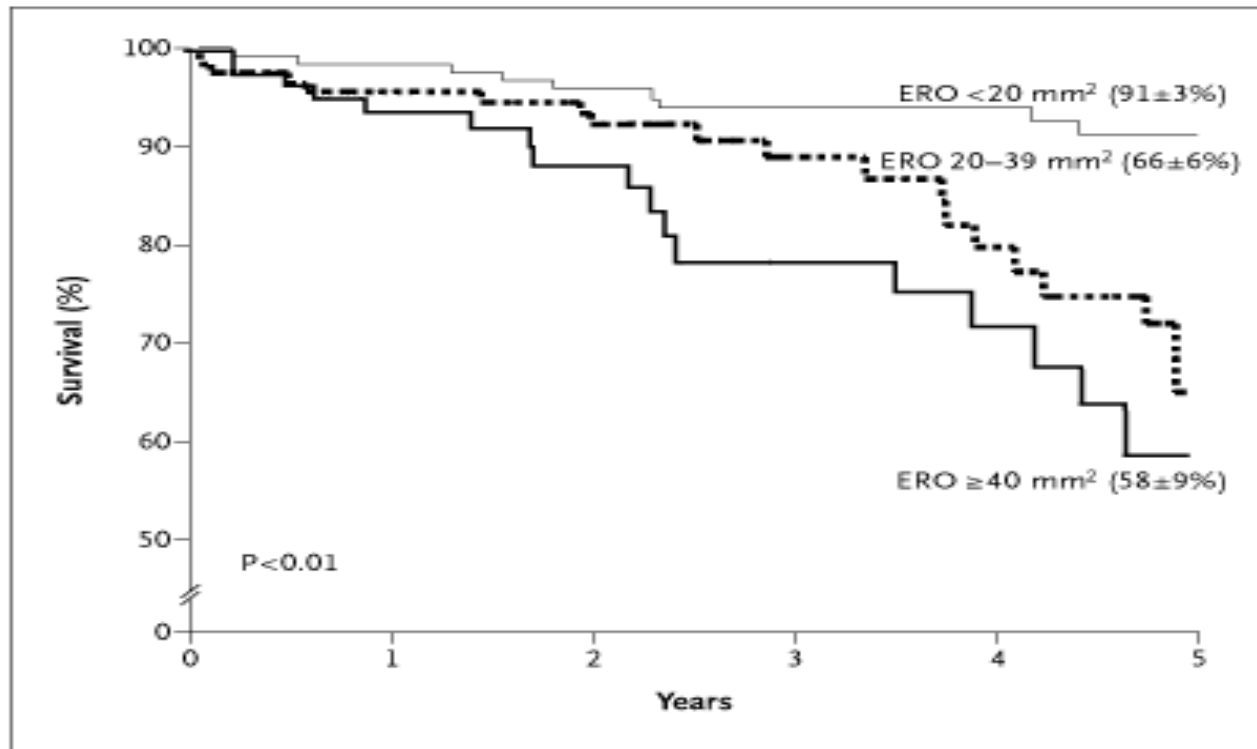
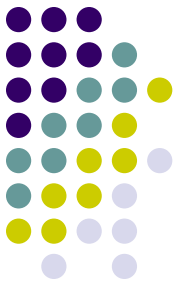


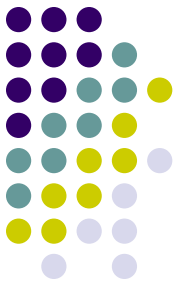
Early surgery for flail mitral leaflet improves outcome Early surgery for flail mitral leaflet improves outcomes as compared to conservative therapy. Among 221 with mitral regurgitation due to a flail leaflet who were eligible for surgery, 63 underwent early mitral valve surgery while 158 were treated medically. Compared to medical therapy, early surgery was associated with a higher overall survival ($p = 0.028$), lower cardiac mortality ($p = 0.025$), a lower incidence of atrial fibrillation ($p = 0.032$), and a lower incidence of heart failure ($p = 0.046$). (Redrawn from Ling, LH, Enriquez-Sarano, M, Seward, JB, et al, *Circulation* 1997; 96:1819.)



Survival after mitral valve surgery is better for patients in NYHA class I/II independent of LVEF Long-term patient survival after mitral valve surgery for chronic mitral regurgitation is better in those in NYHA class I/II compared to those in class III/IV at the time of surgery. The benefit is independent of the left ventricular ejection fraction (LVEF). (Data from Tribouilloy, CM, Enriquez-Sarano, M, Schaff, HV, et al, *Circulation* 1999; 99:400).

Quantitative Determination of outcomes in asymptomatic MR

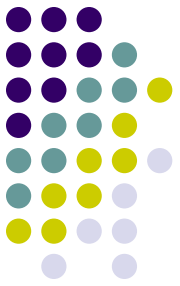




Mitral Valve surgery

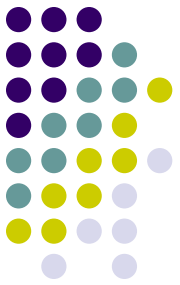
1. Mechanical valve ie St. Jude.
Advantage: longevity. Problem: Warfarin
2. Tissue valve. Advantage: no Warfarin. Problem: longevity
3. MV repair. Advantage: better LV function , no Warfarin, problem: may not be technically feasible.

Indications for Surgery for MR



1. Preop LV function is best predictor of long term morbidity and mortality
2. Early surgery for severe MR is key.
3. Recommendations for surgery
 - Any pt. with severe MR even if no symptoms, but good LV function, as long as
 - Low op risk and chance for valve repair
 - Asymptomatic patients with NL EF but effective regurgitant orifice by Doppler is 40 mm squared or more.

Functional anatomy and outcome of MR surgery



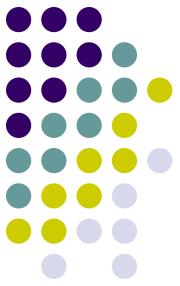
- TTE and particularly TEE may be very helpful in deciding which valves can be repaired.
- Patients with floppy valves are often good candidates for repair.
- Very calcified valves, ischemic MR and secondary (functional) MR patients are usually not candidates for repair.



Indications for Surgery in MR

1. Consider waiting for signs of decreased LV FX or progressive dilatation in pts with:
 - Less than severe MR
 - High OP risk due to co-morbidities
 - Likelihood of repair low
 - Narcotic addicts with endocarditis

Why choose MV Repair over MVR

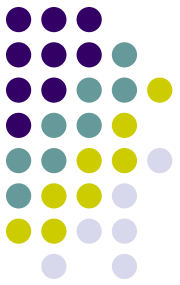


1. Lower OP mortality
2. Improved long-term survival
3. Better preserved LV function
4. Lower incidence of thromboembolism, bleeding and endocarditis

Why is MV repair gaining popularity in US

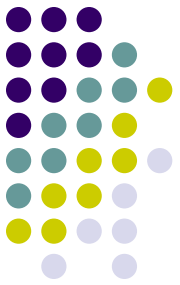


- Improved surgical techniques and intraop TEE
- Dominant pathology is degenerative disease, more repairable
 1. Factors which ↓ likelihood of repair are
- Annular calcification, RHD and anterior leaflet dysfunction



Causes of MV Repair Failure

1. Valve related 65%
 - Fibrotic/calcified leaflet or annulus
 - New perforated leaflets or ruptured chordae
 - Procedure related 35%
 - Failure of chordae/leaflet repair or failure of ring annuloplasty



MV Repair Summary

1. Valve repair techniques can be used on many patients with MR
2. Repair must be tailored for the individual valve pathology
3. Operative mortality is low, particularly with low risk patients
4. Early surgical intervention gives the best long term results
5. Several catheter techniques are being developed for repair of MV

Chronic Severe Mitral Regurgitation

